Southwestern

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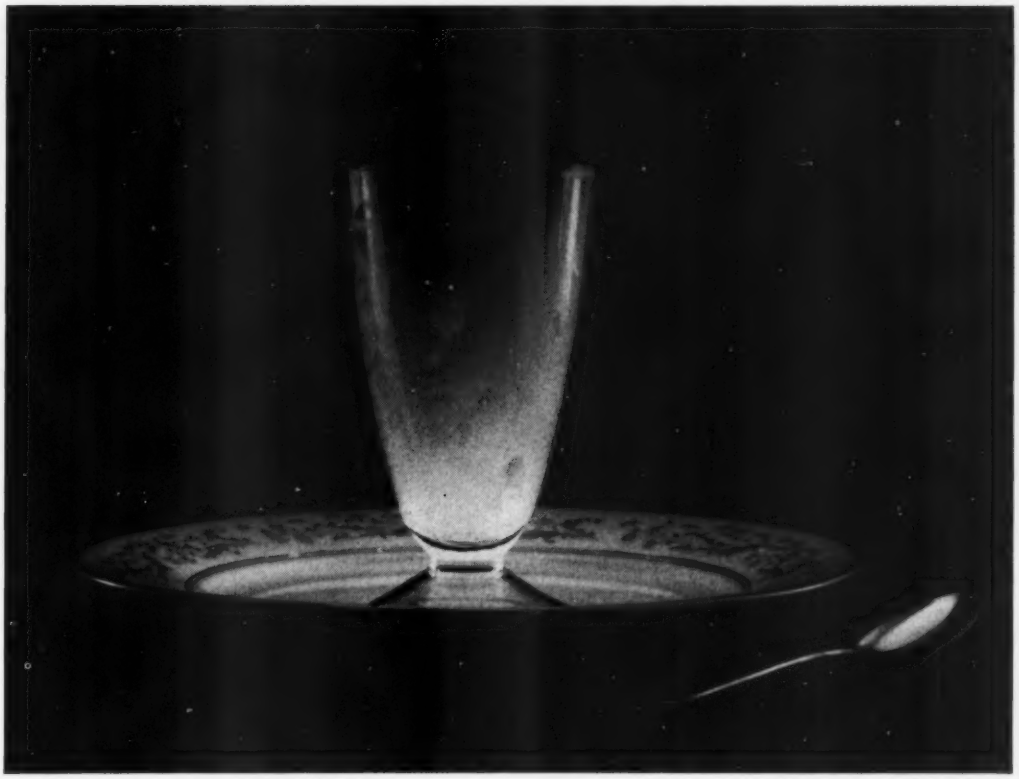
References

1. McCarthy, C. G., and Finland, M.: Absorption and Excretion of Four Penicillins, New England J. Med., *263*:315, 1960. 2. McCarthy, C. G., Hirsch, H. A., and Finland, M.: Serum Levels after Single Oral Doses of 6-(α-phenoxypropionamido) Penicillanate and Penicillin V, Proc. Soc. Exper. Biol. & Med., *103*:177, 1960. 3. Griffith, R. S.: Comparison of Antibiotic Activity in Sera after the Administration of Three Different Penicillins, Antibiotic Med. & Clin. Therapy, *7*:129, 1960. 4. Editorial: New England J. Med., *263*: 361, 1960. 5. Editorial: New York J. Med., *60*:498, 1960.



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COMING MEETINGS

Texas Chapter, American College of Chest Physicians, Annual Meeting, Moody Convention Center, Galveston, April 23, 1961.

Texas Orthopaedic Association, Annual Meeting, Galveston, Texas, April 24, 1961.

Arizona Medical Association, 70th Annual Meeting, Scottsdale, Arizona, April 26-29, 1961.

New Mexico Medical Society, 79th Annual Meeting, La Fonda Hotel, Santa Fe, May 17-20, 1961.

United States-Mexico Border Public Health Association, Annual Meeting, San Diego, June 25-29, 1961.

Postgraduate Course in Pedriatics, The University of Colorado School of Medicine, Stanley Hotel, Estes Park, Colorado, August 21-25, 1961.

Western Association of Railway Surgeons, Annual Meeting, Holiday Hotel, Reno, Nev., Sept. 28-30, 1961.

Southwest Obstetrical & Gynecological Society, Eleventh Annual Meeting, Konakai Club, Sna Diego, Oct. 15-17, 1961.

Southwestern Medical Association, 43rd Annual Meeting, Tropicana Hotel, Las Vegas, Nev., Oct. 19-21, 1961.

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eferences:
1. Graham, W.: Canad. M.A.J. 82:1005 (May 14) 1960. 2. Vaughn, P. P.; Howell, D. S., and Kiem, I. M.: rth. and Rheumat. 2:212, 1959. 3. O'Reilly, J.: J. Irish M.A. 43:106, 1960. 4. Connell, J. F., Jr., and Rousselot, L. M.: Am. J. Surg. 98:31, 1959. 5. Brodie, B. B., et al., in Contemporary Rheumatology 1956, p. 600. 6. Stein, I. D.: nn. N. Y. Acad. Sc. 86:307 (March 30) 1960. 7. Barczyk, W., and Röth, W.: Praxis 49:530, 1960. 8. Miller, L. M., et al.: Antibiotic Med. and Clin. Rousselot, L. M.: Am. J. Surg. 97:429, 1959. 10. Summary of individual case histories submitted to Geigy. 11. Domenjoz, R.: Ann. N. Y. Acad. Sc. 86:283, 1960. 12. Smyth, C. J.: Ann. N. Y. Acad. Sc. 86:292, 1960.

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New Mexico Medical Society To Meet in Santa Fe, May 17-20

The 79th annual meeting of the New Mexico Medical Society will be held May 17-20, 1961, in Santa Fe, N. M., with headquarters in the La Fonda Hotel.

Speakers will be Dr. Edwin L. Kendig, Richmond, Va., Associate Professor of Pediatrics and Director of the Child Chest Clinic at the Medical College of Virginia; Dr. Vincent P. Collins, Professor of Radiology and Chairman of the Department at the Baylor College of Medicine, Houston; Dr. John W. Pender, Palo Alto, Calif., head of the Department of Anesthesiology at the Palo Alto Medical Clinic and Clinical Assistant Professor of Anesthesiology at Stanford University; Dr. Marvin A. Block, Buffalo, Assistant Clinical Professor of Medicine at the University of Buffalo School of Medicine and Chairman of the AMA's Committee on Alcoholism;

Dr. Ben Eiseman, Professor of Surgery at the University of Colorado and Chief of Surgical Service at the Veterans Hospital in Denver; Dr. Edgar S. Gordon, Madison, Professor of Medicine at the University of Wisconsin; Dr. Robert H. Barter, Professor and Department Head of Obstetrics and Gynecology at the George Washington University School of Medicine in Washington, D. C.; Dr. Horace E. Campbell, Denver, Chairman of the Automotive Safety Sub-Committee of the Colorado State Medical Society and member of the Colorado Citizens Traffic Safety Committee; Dr. John S. Chapman, Assistant Dean and Professor of Medicine at Southwestern Medical School at Dallas; and John H. Furbay, Ph.D., New York, Director of Trans World Airlines' World-wide Education Program; General Albert Schwichtenberg, M. C. (retired), Albuquerque, Lovelace Foundation; Col. S. W. Cavender, M. C., Albuquerque, Sandia Base; and Dr. North Longfield, Albuquerque, Veterans Hospital.

The complete program is as follows:

PROGRAM

Wednesday, May 17
General Meeting
Santa Fe Room, La Fonda

Presiding: Lewis M. Overton, M.D., Albuquerque, Immediate Past President, New Mexico Medical Society

1:30 p.m. Invocation
Sarah Bowen, M.D., Santa Fe
Welcome
Honorable Leo Murphy, Mayor, City of Santa Fe
Welcome
Fred Soldow, M.D., Santa Fe, President, Santa Fe County Medical Society

1:45 p.m. Presidential Address
Allan L. Haynes, M.D., Clovis, President, New Mexico Medical Society

First Clinical Session

Presiding: Allan L. Haynes, M.D.

2:15 p.m. Peptic Ulcer Co-existing with Chronic Pulmonary and Liver Disease; Clinical and Theoretic Implications
Ben Eiseman, M.D., Denver

3:00 p.m. Nerve Entrapment Syndrome
Clinton Morgan, M.D., Albuquerque

3:15 p.m. Visit Exhibits

3:30 p.m. Clinical Pathological Conference
Moderator: William Hentel, M.D., Albuquerque
Participants: Edgar S. Gordon, M.D., Madison, Wis.; Vincent Collins M.D., Houston; Ben Eiseman, M.D., Denver; and Harry Ellis, M.D., Santa Fe

6:30 p.m. Cocktails

7:30 p.m. Specialty Group Dinners

Thursday, May 18
Second Clinical Session

Presiding: William Badger, M.D., Hobbs, President-Elect, New Mexico Medical Society

9:00 a.m. Lipid Metabolism and Atherosclerosis
Edgar S. Gordon, M.D.

9:45 a.m. The Internal Cardiac Pacemaker in the Treatment of Stokes-Adams Syndrome
Alan L. Frankel, M.D., Albuquerque

10:00 a.m. The Duration of Cancer Prior to Diagnosis
Vincent P. Collins, M.D.

10:45 a.m. Visit Exhibits

11:15 a.m. Panel: Pancreatitis
Moderator: Andrew M. Babey, M.D., Las Cruces
Participants: Edgar S. Gordon, M.D., Vincent P. Collins, M.D., Ben Eiseman, M.D., and Peter Van Schoonhoven, M.D., Albuquerque
Afternoon Free

6:30 p.m. Dinner Dance

Friday, May 19
Third Clinical Session

Presiding: R. C. Derbyshire, M.D., Santa Fe, Vice-President, New Mexico Medical Society

9:00 a.m. Unclassified Mycobacteria in Children
Edwin L. Kendig, M.D., Richmond, Virginia

9:45 a.m. Unusual Varieties of Obstetrical Hemorrhage
Randolph V. Seligman, M.D., Albuquerque

10:00 a.m. Ectopic Pregnancy; A Constant Diagnostic Problem
Robert H. Barter, M.D., Washington, D. C.

10:45 a.m. Visit Exhibits

11:15 a.m. Monitoring During Anesthesia
John W. Pender, M.D., Palo Alto, Calif.

Group Clinical Session A
Trauma and Disaster Planning
Symposium on Trauma

Co-Chairmen: Harold Fenner, M.D., Hobbs, Chairman, Accident Prevention Committee, New Mexico Medical Society
General Albert Swichtenberg, MC (retired), Lovelace Foundation, Albuquerque, Chairman, Disaster Planning Committee

2:00 p.m. General Principles of Shock
John W. Pender, M.D., Palo Alto, Calif.

2:30 p.m. Orthopedic Traumatic Problems
John F. Boyd, M.D., Albuquerque

3:00 p.m. We Are Entitled to Modern Crash Protection in Our Automobiles
Horace E. Campbell, M.D., Denver

3:45 p.m. Coffee

Panel on Disaster Planning

4:00 p.m. Step by Step Approach to General Disaster Planning
Col. S. W. Cavender, MC, USA, Sandia Base, Albuquerque.

4:45 p.m. Bernalillo County Medical Association's Approach to Disaster Planning
General Albert Schwichtenberg

Group Clinical Session B
Alcoholism

Presiding: Richard Angle, M.D., Santa Fe, Chairman, Program Committee

2:00 p.m. General Aspects of the Problem of Alchoholism
Marvin A. Block, M.D., Buffalo

3:00 p.m. Psychiatric Aspects of the Problem of Alcoholism
William F. Sears, M.D., Los Alamos, Chairman, Mental Health and Alcoholism Committee

3:30 p.m. Coffee

3:45 p.m. The New Mexico Situation Regarding Alcoholism
Warren Brown, M.D., Albuquerque, Chairman, Governor's Special Committee on Mental Health

4:15 p.m. Panel Discussion
Moderator: Warren Brown, M.D., Albuquerque
Participants: Marvin A. Block, M.D., Buffalo; Charles A. Beeson, M.D., Albuquerque; Earl Latimer, M.D., Roswell

6:30 p.m. Specialty Group Dinners

Saturday, May 20
Symposium on Tuberculosis

Co-Sponsors: Public Health Committee, New Mexico Medical Society and New Mexico Public Health Department

Co-Chairmen: Roy F. Goddard, M.D., Albuquerque, Chairman, Public Health Committee, New Mexico Medical Society; Stanley Leland, M.D., Santa Fe, Director, New Mexico Department of Public Health

The Medical Aspects of Tuberculosis

9:00 a.m. Tuberculosis in Childhood
Edwin L. Kendig, M.D., Richmond, Virgilnia

10:00 a.m. Adult Tuberculosis
Sumner Cohen, M.D., Oak Terrace, Minn.

11:00 a.m. TBC-Like Pulmonary Syndromes
John Chapman, M.D., Dallas

12:15 p.m. Luncheon
Speakers: Joseph Gordon, M.D., Albuquerque, President, New Mexico Tuberculosis Association; North Longfield, M.D., Albuquerque, President, New Mexico Thoracic Society; J. E. J. Harris, M.D., Albuquerque, President, New Mexico Chapter, A.C.C.P.

The Socio-Economic Aspects of Tuberculosis

2:00 p.m. The Socio-Economic Aspects of Tuberculosis
Mrs. Ruth Taylor, New York, Representative, National Tuberculosis Association

3:00 p.m. Joint Report of the Public Health Committee and the New Mexico Tuberculosis Coordinating Council
Hugh B. Woodward, M.D., Albuquerque

4:30 p.m. Panel on New Mexico Tuberculosis Report
Moderator: Stanley J. Leland, M.D.
Participants: Edwin L. Kendig, M.D.; Sumner Cohen, M.D.; John Chapman, M.D.; Mrs. Ruth Taylor

Auxiliary Program
Wednesday, May 17

9:00 a.m. to **11:30 a.m.** Hospitality Coffee

11:00 a.m. Executive Meeting

Thursday, May 18

9:30 a.m. Delegates Meeting

12:30 p.m. Luncheon
Speaker: Mrs. Harlan English, Danville, Ill., President-Elect, Auxiliary to the American Medical Association

2:00 p.m. Executive Meeting

The Woman's Auxiliary to the Santa Fe County Medical Society will offer personally conducted tours of Artist's Studios, Museums and Shops of Santa Fe. Arrangements for the various tours can be made at the Auxiliary's registration desk and will be available on Wednesday, Thursday and Friday.

A Review of Infant Mortality in New Mexico and the Bordering Mexican States*†

(Section I)

ROY F. GODDARD, M.D.[1], *Albuquerque*
STANLEY J. LELAND, M.D.[2], *Santa Fe*
JOHN C. COBB, M.D.[3], *Baltimore*

1. Director, Pediatric Research Department, Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico; Consultant in Problems of the Newborn to the New Mexico Department of Public Health; Pediatric Consultant, Navajo Medical Center, Ft. Defiance, Arizona; Chairman, Public Health Committee, New Mexico Medical Society.
2. Director, New Mexico Department of Public Health, Santa Fe, New Mexico.
3. Assistant Professor, School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Md. (present address: University of Punjab, Lahore, Pakistan); formerly assigned to the Maternal and Child Health Division, United States Public Health Service, Division of Indian Health, Albuquerque Area Office, Albuquerque, New Mexico.

Infant Mortality Rates in New Mexico and the Bordering Mexican States

Introduction

Infant mortality today remains one of the gravest problems facing the states that comprise the membership of this organization, the United States-Mexico Border Public Health Association. Infants dying under the age of one year constitute approximately one-tenth of the total number of deaths from all causes in the United States.[1] The rate is even higher in many countries, and throughout the world, there is no chart of the ten principal causes of death which does not list this problem as one of its most significant.

A comparison of birth rates and neonatal mortality rates of the United States, the state of New Mexico, and Mexico reveals the birth rate to be 25 per 1000 population for the United States, 34 for New Mexico and 45 for Mexico (see fig. 1). The infant mortality rate under the age of one year for the year 1958 was 27 per 1000 live births for the United States, 37 for New Mexico, and 81 for Mexico.[2] The implication that a higher birth rate may influence the infant mortality rate exists. Comparative birth rates and infant mortality rates for the Mexican border states are presented in Tables one and two.[3]

Infant Mortality in New Mexico

We believe that many of the problems that exist in New Mexico today also exist in old Mexico and we, therefore, feel that enlarging on our specific problem in New Mexico may be of value to our sister states along the border.

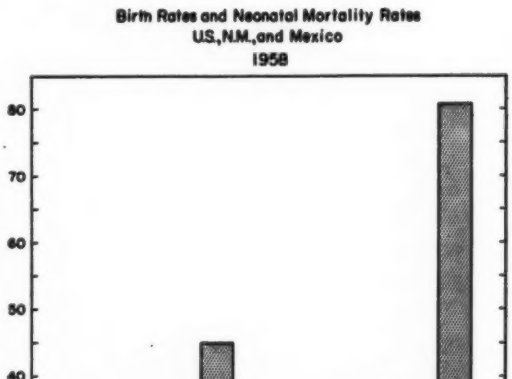

Figure 1

*Presented at the 18th Annual Meeting of the United States-Mexico Border Public Health Association, before the Maternal and Child Health Section, Hermosillo, Sonora, Mexico, April, 1960.

†To be published in three sections in successive issues of SOUTHWESTERN MEDICINE.

For the past several decades New Mexico has had the highest infant mortality rate of any of the 48 states in the United States. (The figures per 1000 live births for the United States, New Mexico, Alaska and Hawaii in 1957 were 26.3, 39.4, 38.0 and 24.0 respectively.)[4]

Figure two, however, shows the progressive decline in infant mortality in the United States and New Mexico from 1944 through 1959, a period of the last 15 years. There has been a drop in the United States from 1944 to 1948, which has remained essentially stationary. The rate for New Mexico in 1945 was 100. This has decreased progressively, step-wise, until in 1959, this past year, we had our lowest mortality rate of 33.4 for the state as a whole*.[5] We think that many of the programs we have instituted have influenced this progressive drop in mortality rate.

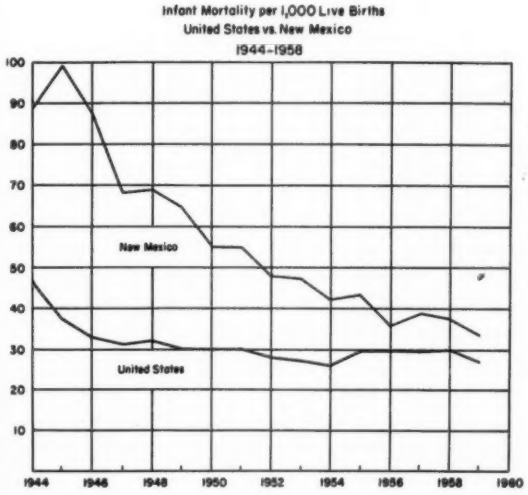

Figure 2

There are many factors which contribute to New Mexico's high infant mortality rate. New Mexico today remains a tricultural state in background, influenced by the Spanish culture and settlement; that of our precursors, the Indians; and our most recent additions the Anglo and other cultural backgrounds, which today constitute our largest group in the state.

If we analyze our infant mortality by races,

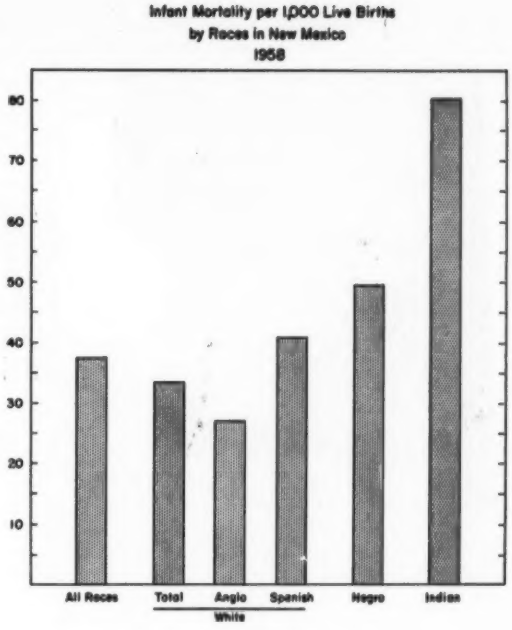

Figure 3

Figure three shows the infant mortality for the state as a whole in 1958 to be 37.4 per 1000 live births; that for the white population, combining all whites, gave a total of 33.4; 27.3 for the Anglo element, and 41 for those of Spanish surname. The mortality rate for Negroes was 49.6 and that for the Indian population 80.3.[6] This is not to infer that the cultural aspect is the most significant in this differentiation of infant mortality by races.

Figure four is a map of the State of New Mexico with its 32 counties. San Juan and McKinley Counties which are in the northwestern part of the state had a high infant mortality rate, between 80 and 100, per 1000 live births in the years 1950-54.

Another area in the state, midcentrally located, San Miguel County, had a similar high rate. Socorro, Mora, Sandoval, Taos, and DeBaca also had high infant mortality rates. Other counties, such as Los Alamos, Curry, Roosevelt, and Lea, had less than 30 deaths per 1000 live births, and Bernalillo County had 30-39.

In figure five, the infant mortality per 1000 live births in selected counties of New Mexico has been plotted. Lea County had the lowest infant mortality rate in the state with 21.4 per 1000 live

*In 1959 New Mexico ranked seventh among the 50 states of the United States.

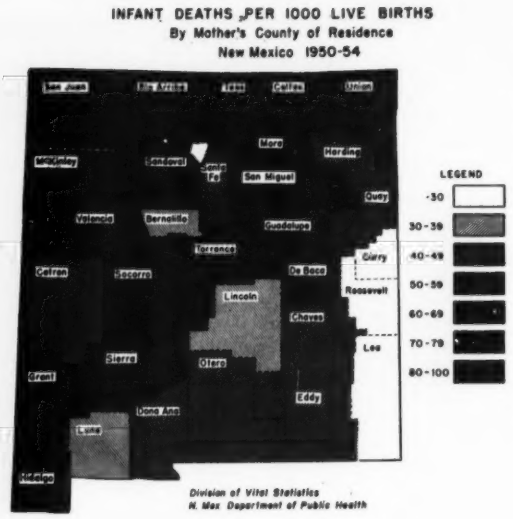

Figure 4

births; Bernalillo County had an intermediate rate, 31.7; McKinley County of the northwestern part of the state had 56.7; and San Miguel County, the highest, had a rate of 57.5. There are certain factors which contribute to the differences in infant mortality in these various counties.[7]

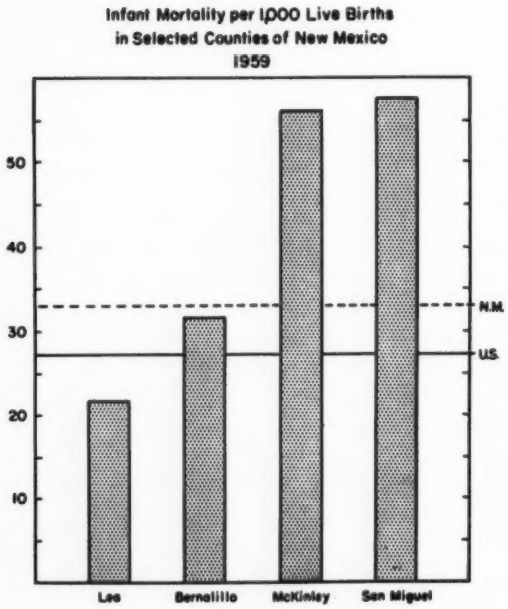

Figure 5

Figure six shows some of these factors. First, compare the 1955 population in these various counties. In Lea and Bernalillo Counties there has been a sizable increase in population; about half this increase in McKinley County, and a decrease of 11 per cent in the population of San Miguel County in this 5-year period.

The relationship of doctors per 1000 population is 0.3 in McKinley and Lea; and 0.5 in San Miguel; and 1.0 in Bernalillo County. The birth rate per 1000 population is low in Lea County and San Miguel County and is relatively high in McKinley and Bernalillo Counties. The infant mortality rate is quite high in McKinley and San Miguel Counties.

The number of premature births ranges from nine to 12 in all counties. As to population by races, Lea County is predominately Anglo; Bernalillo County about two-thirds Anglo; in McKinley County 62 per cent or almost two-thirds are of Indian extraction; and in San Miguel County 77 per cent are of Spanish surname.

The population with an income of less than $500 per year is over 25 per cent in McKinley and San Miguel Counties; the population with schooling of less than eight years is over 50 per cent in both of these counties; the number of dwelling units with no running water is 40 per cent in both of these counties; the number of dwelling units with no inside toilet is 63 per cent in both of these counties; and the per cent of dwelling units with more than one and a half persons per room is 30 per cent and 44 per cent in these counties, respectively.[8]

Another significant factor in San Miguel County is the high per cent of infants born out of wedlock, 34 per cent. This, we believe, shows some of the factors relating cultural, social and economic influences in these selected counties in New Mexico.

In Figure seven causes of infant death for the State of New Mexico in 1954 and 1958 have been graphed, together with figures for 1959.[9] Of the total number of deaths, over 20 per cent of these are attributed to immaturity, and the majority of these deaths occur under the age of 28 days.

Immaturity as a cause has decreased slowly in

SOME FACTORS INFLUENCING THE INFANT MORTALITY RATE IN SPECIFIC COUNTIES IN NEW MEXICO

	Mortality Rate 75-100		Mortality Rate 30-40	
1955 Population	McKinley 33,800	San Miguel 23,700	Lea 50,206	Bernalillo 205,500
Per cent Change 1950-55	23	11	63	44
1955 Doctors per 1000 Population	0.3	0.5	0.3	1.0
1955 Birth Rate/1000 pop.	36	14	15	34
1955 Infant Mortality (per 1000 live births)	75	96	39	33
1955 Premature Births (per cent of live births)	10	12	9	11
1950 Population				
% Anglo	23	23	98	68
% Spanish surnames	15	77	2	30
% Other Races (pred. Indian)	62	--	--	2
% Population with income less than $500/yr	27	27	8	11
% Population over 25 yrs with less than 8 years schooling	49	54	22	22
% Dwelling units with no running water	42	36	6	10
% Dwelling units with no inside toilet	63	63	24	27
% Dwelling units with more than 1.5 persons per room	44	30	15	14
% Infants born out of wedlock	Unknown	34	16	Unknown

N. M. Dept. Vital Statistics
Welfare Invest. Com.
U N M Bus. Res. Bureau

Figure 6

1958 from 22 to 21 and in 1959 to 20½. Probably the next most significant cause of death during this first 28-day period is atelectasis and asphyxia which in 1954 and 1958 was around 10 per cent of the total number of deaths and this rose in 1959 to 13 per cent, presumably because more babies were signed out as atelectasis and asphyxia in the ensuing years.

The next most significant factor in the neonatal period was birth injuries, and they increased from six to eight in 1954 and 1958 to nine in 1959. Next are congenital malformations which have risen from seven to 10 to 11, respectively, in 1954, 1958, and 1959. However, deaths after the age of 28 days are considerable in this category. At least one-third of the total number of deaths occur after the age of 28 days.

Pneumonia is probobly the next most significant factor in the immediate neonatal period, and this as a total cause of death has remained around 13 for all three years. Again about two-thirds of these deaths, ascribed to pneumonia, occurred after the 28-day period. Erythoblastosis remains around one to 1.6 for all three years.

Miscellaneous causes, including other specific causes and unknown ill-defined causes, constitute about 20 per cent of the total number of deaths. Again, a larger portion of these occur after the age of 28 days. The most significant cause of death over the age of 28 days has been diarrhea and dysentery for many years. Considerable progress has been made during the last five years, with the rate falling from 14 to 11 and in 1959 to six and one-half. This shows considerable improvement in the causes of deaths in our older infants, and we hope that this trend will continue in the State of New Mexico.

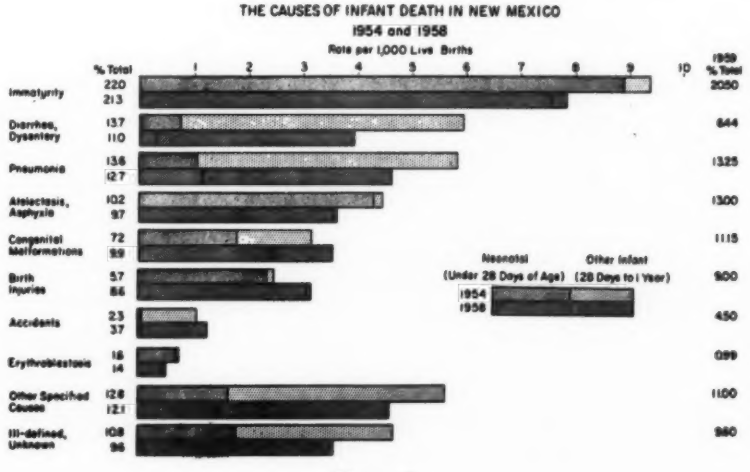

Figure 7

TABLE I

BIRTH RATE IN THE MEXICAN STATES BORDERING THE UNITED STATES

1954-1957

STATE	1954	1955	1956	1957
Baja California N.	51.2	51.6	48.9	49.5
Baja California S.	44.4	46.0	45.7	46.3
Sonora	55.4	54.4	56.3	57.3
Chihuahua	45.7	45.0	45.4	47.9
Coahuila	47.2	49.4	47.1	47.3
Nuevo León	44.5	45.1	45.6	48.0
Tamaulipas	49.0	46.8	45.4	44.2
Mexico as a whole	46.4	46.4	46.8	46.9

Note—Rates per 1000 inhabitants estimated June 30 of each year.
(From Bureau of Vital Statistics, Mexico, D.F.)

TABLE II

INFANT MORTALITY IN THE MEXICAN STATES BORDERING THE UNITED STATES

1954-56

STATE	1954	1955	1956
Baja California N.	79.7	71.3	67.9
Baja California S.	73.4	60.5	82.7
Sonora	75.2	72.7	63.8
Chihuahua	92.2	89.5	79.5
Coahuila	82.2	79.7	68.7
Nuevo León	71.4	73.4	53.2
Tamaulipas	54.7	67.6	55.3
Mexico as a whole	80.5	83.3	71.0

Note—Rates per 1000 registered live births.
(From Bureau of Vital Statistics, Mexico, D.F.)

This has been a significant factor in many of the counties that we have talked about previously in this paper and the Public Health and Indian Service are working together with the private practitioners of medicine to eradicate diarrhea and dysentery as an outstanding cause of death over the age of 28 days of life. The trend from Figure seven shows that immaturity is being used less as a diagnosis, asphyxia and atelectasis more, that there is a rise in causes of death in congenital malformations and birth injuries, and a decrease in some of the other causes of infant death.*

*Principal causes of infant mortality in Mexico for the years 1955-57 were: diseases of early infancy 37.4% of total causes of death, gastroenteritis 21.0%, influenza and pneumonia 20.8%, and congenital malformations 2.5%.[10]

Postgraduate Course to Be Presented

Pediatric Allergy is the subject of a one day course to be given by the El Paso Division of the University of Texas Postgraduate School of Medicine Sunday, May 21, 1961, in the El Paso County Medical Society's Turner Home at 1301 Montana Avenue in El Paso.

Dr. J. Leighton Green, director, has announced that the Texas Academy of General Practice has approved the course for Category I credit.

Arthritis

Biochemical Suffocation

R. P. WATTERSON, M.D., *Scottsdale, Ariz.*

Although not in general use, knowledge of the cause of arthritis exists, as well as measures necessary for its prevention and treatment. It is the obligation of the medical profession to recognize and use this information. Such measures consist simply of a sound biochemical approach to the problem and the elimination of adrenal steroid therapy as hereinafter explained.

Since it is already generally accepted that pain, fatigue and dysfunction are directly resultant from anoxia, it is practical to examine the physiological manifestations of anoxia. The signs, symptoms and pathologic changes are the same whether the cause of the anoxia is mechanical or biochemical.

Anoxia
Mechanical Biochemical

Fatigue	Reversible
Dysfunction	Reversible
Pain	Reversible
Atrophy	Reversible
Fibrosis	Reversible
Death	Irreversible

The majority of clinicians are aware of the relationship between oxygen supply and the changes in structure and function, but most have failed to correlate the known biochemical data, recognized pathology and physiologic response.

Results of Anoxia

Recognized as being the result of anoxia are: complaints of fatigue and decreased endurance at high altitudes and with cardiac failure, emphysema and asthma; the cerebral dysfunction with senile arteriosclerosis; the anoxic distress of angina pectoris and intermittent claudication; the atrophic deformity of Volkmann's contracture (presenting atrophy and secondary fibrosis from prolonged mechanical anoxia); and the sclerodermatous changes about the ankles of patients with long-standing varicosities.

We are familiar with the inactivation of the cytochrome enzymes with cyanide, the carbon monoxide inactivation of hemoglobin and the formation of methemoglobin with exposure to nitrites, the resultant death being nothing more or less than biochemical suffocation.

Regardless of specific diagnosis, the following conclusions appear indicated:

1. Persistent fatigue is collateral with decreasing cellular oxidative capacity.

2. Acute or chronic anoxia augments existing dysfunction, regardless of the system involved.

3. The role of anoxia in atrophy of tissue must be considered before the inquiry is complete.

4. Fibroplasia is the direct expression of prolonged anoxia.

5. Nature induces a regional anoxia to stimulate the repair-healing-dehydration mechanism.

6. Tissue death, local or general, is always due to anoxia, however incited.

Factors

Factors common to all forms of arthritis are: hyalinization of connective tissue, pain, progressive accumulation of fibrous tissue and disturbance of joint morphology.[1-11] Often these factors go unnoticed until the pain or decrease in mobility become acute. The physician can employ measures which will arrest the progressive fibroplasia and produce gradual mobilization and metabolism or excretion of the accumulated pathologic tissues. Such measures include nutritional guidance, increase in magnesium and trace mineral ingestion, reduction of exposure to toxic chemicals and heavy metals such as lead, and supplementation with etioporphyrin, type III, to facilitate synthesis and maintain adequate titers of heme-derived enzymes.

Physicians should consider the known relationships between the porphyrinic enzymes, oxidative mechanisms and the total functional capacity of the individual patient. Failure of medical educators to disseminate this information is inexcusable. Without this knowledge physicians cannot intelligently prescribe preventive or curative schedules.

Treatment

The adrenal atrophy which accompanies corticosteroid therapy undermines the entire biochemical stability of the patient. The observed complications of hypertension, psychoses, visceral perforation, skeletal demineralization and probable reduction of resistance to carcinogenesis con-

traindicates the use of corticosteroids in any disease.

Elimination of corticosteroids necessitates a total program effective for treatment and prevention. The patient must be instructed to maintain a long-range program to improve his physical fitness. It is most effective if it includes reduction of intake of magnesium-low and mineral-low foods (refined sugar, karo and white flour); reduction of lead intake by elimination of tea, aluminum cookware and tobacco; elimination of milk to withdraw growth-stimulating factors and the possibility of viral seeding; substitution of vegetable oils and butter to replace hydrogenated fats, thus increasing the intake of linoleic acid; the maintenance of vitamin E intake to insure the antioxidant activity provided by the tocopherols. A long-range demand for non-essential yet caloricallyvaluable tissue components must be created by reducing total food intake[12-25].

Following is an initial diet recommended for new patients, found satisfactory for the majority. Avoidance of foods to which the patient is specifically allergic is recommended.

Breakfast: Meat, any type, steak preferred, to include some fat; Eggs; Vegetable juice; Postum, Sanka or black coffee.

Lunch: Fish, fowl, seafood, or buttermilk; Chef's type salad, oil-vinegar dressing; soup; one or two yellow or green vegetables seasoned with salt, butter, cayenne red pepper.

Dinner: Salad, any type dressing; One green or yellow vegetable; Fresh fruit or melon; Brown rice or cooked cereal, not to exceed three times weekly.

Progressive exercise schedules are mandatory and must be maintained to increase joint mobility, flexibility of the spine and increase muscular strength and endurance.

Therapeutic

Item	*Comment*
*Etioporphyrin, Type III	Hemoproteins are etioporphyrins, type III[27]. Functional improvement and increase in tissue metalloporphyrins is accelerated by supplementation with utilizable porphyrin (Kosaki)[28].
**Magnesium	Supplementation with absorbable magnesium chelates provides magnesium for the metabolism of non-essential tissues and mobilization of calcium from soft tissue deposits.
B-Complex Vitamins	Israel[26], reporting a ten-year study, documents the benefits derived by thyroid, B-vitamins and lipotropics in arthritis.
Thyroid	Thyroid extract or iodized threonine preparations enhance intracellular oxidation and proves beneficial in proper dosage in all arthritics.

Patients who have received corticosteroids as part of their previous medication are salt losers because of inadequate corticosteroid titer. As much as one to two teaspoons daily may be found necessary to maintain adequate NaCl serum levels. Temporary intake of up to 10,000 mg. of ascorbic acid daily may be necessary to resaturate these individuals and reestablish capillary integrity. These measures are applied to osteoarthritis, rheumatoid and gouty alike, since they are directed toward a reduction in stroma and an increase in parenchyma.

The specific use of uricosuric agents and colchicine in gouty arthritis needs no discussion. Patients having a history of lead poisoning or excessive smoking and those with porphyrinuria benefit from EDTA taken over a period of three to five months. Ice packs applied to painful hot joints and bursitis are indicated instead of heat. Estrogenic and androgenic hormone supplementation is advisable where anabolic mechanisms can be stabilized with this addition to the treatment schedule.

Clinical Evaluation

Clinical evaluation of the aforementioned factors in treatment has been under investigation in my office for the past twelve years. These various factors have been controlled singly and in combination to gain impressions as to the validity of the concept that they are essential parts of a program designed to promote total physical fitness.

For the past five years, more than fifty per cent of my practice has been devoted to the care of arthritics. The long-range clinical course of these patients has convinced me that arthritis need not be a chronic disease if the patient has sufficient fortitude and self-discipline to maintain a total program guided by the foregoing recommendations.

During the past three years, 250 patients have been closely observed on the specific therapy as outlined. The following results are characteristic.

Grade I rheumatoid arthritics usually become asymptomatic within two months requiring minimal medication; grades II and III rheumatoid arthritics, who have not had previous corticosteroid therapy, show progressive ameliorization of their disease during periods ranging from four months to a year.

The majority of grade III and IV rheumatoid arthritics seen have had previous corticosteroid therapy. The deterioration produced by this treatment is a major factor in delaying and sometimes making impossible the return to a state of health. When joint destruction has occurred, prosthetic surgery or fusion is often desirable.

Improvement in this group is markedly delayed and often completely inhibited if the patient will not cease smoking. In the same group, when excessive waterlogging occurs, it is sometimes necessary to instruct the patient in the practice of short fasts extending from four to ten days.

Withdrawal of collagen and fibrous tissue components is evidenced by the contouring of joints, decrease in size and number of subcutaneous nodules, improved peripheral circulation, increased flexibility of the spine, decrease in the size of Heberden's nodes, decrease in the size of localized fibromata in muscle and improved elasticity of the subcutaneous tissues.

During mobilization of these collagen deposits, regional pain may develop for a short period of time. As the hyalinized material is depleted from the area, these symptoms subside.

Porphyrin supplementation markedly improves arthritics, regardless of classification. These patients show a rapid return to a more normal blood count, a normalization of blood pressure and cholesterol levels, a marked increase in endurance, increased mental alertness and improvement in their total sense of well-being, with a decrease in pain and increased resistance to infection[29-33].

Pain relief in the majority of these patients can be adequately obtained from minimal salicylate therapy. The increased magnesium intake appears to reduce muscle spasm and irritability, and has a definite inhibitory action on the development of acute synovitis. Disappearance of toxic porphyrinuria, which is present in a surprising number of these patients, has also been noted.

Porphyrin and magnesium supplementation can be continued indefinitely; however, the majority of patients, by trial and error, determine when this supplementation can be satisfactorily discontinued, and often request reinstatement of these supplements when symptoms develop. It is my personal opinion that these supplements are indicated until the major quantity of non-essential tissue has been mobilized and metabolized.

The satisfaction of observing progressive patient improvement, after years of chronic disabling illness, will prevent me from ever returning to a strictly symptomatic treatment approach in arthritic disease.

Summary

A concept of arthritis is presented which correlates the symptoms, clinical manifestations and progressive pathologic changes to the mechanisms of oxidative metabolism. Systemic treatment by a total program for improving physical fitness, which includes factors for improving metabolic balance, a rational pattern of nutritional selection with specific magnesium and porphyrin supplementation, is outlined.

The clinical results observed over a period of years in a large number of arthritics in my practice, in which these measures have been employed, are reported. Porphyrin* and magnesium** supplementation is believed to be major factors in the patient's improvement.

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Tumors of the Renal Pelvis*

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Tumors of the renal pelvis and calices are a completely different group from those having their origin in the parenchyma of the kidney. They are closely related to tumors arising in the vesical and ureteral mucosa. Their embryological background is the same and they are identical, histologically.

Renal pelvic tumors are classified by Lowsley and Kirwin as follows:

Epithelial Tumors—Papillary

Papilloma
Papillary epithelioma
Papillary carcinoma (infiltration)

Epithelial Tumors—Non-Papillary

Alveolar or scirrhous carcinoma
Squamous—cell carcinoma

Connective Tissue and Embryonal Tumors (very rare)

Sarcoma
Rhabdomyoma
Myxoma
Angioma
Fibroma
Mixed types

Incidence

Primary tumors of the renal pelvis are rare in comparison with those situated in the parenchyma of the kidney. It is estimated from several comparative studies that only five to seven per cent of all renal tumors occur primarily in the pelvis. Although they are considered to be uncommon, the incidence has grown considerably in the past 25 years.

This is undoubtedly due to increased awareness of these tumors and improved diagnostic methods. They occur usually in middle-aged people, between the ages of 40 to 65, yet they are encountered in the very young and the very elderly.

Men are more often affected than women, the ratio being three to one, and the right kidney is involved slightly more often than the left.

Pathology

The majority of tumors of the renal pelvis are epithelial and arise from the mucous membrane of the pelvis and calices, most of them being of the papillary type. This variety is observed more frequently than the squamous cell type. Relatively 75 per cent of all instances of renal pelvic tumors are papillary.

Although some of the papillomas may be histologically benign it is more practical to consider all papillary tumors of the renal pelvis as malignant tumors and treat them as such because such tumors cannot be safely excised locally or treated by fulgeration. Connective tissue tumors, embryonic and mixed tumors have been reported but they are extremely rare.

Papillomatous tumors of the renal pelvis are of clinical interest because:

(1) Their tendency to occlude the outlet of the renal pelvis with resultant hydronephrosis.

(2) The pressure atrophy of the parenchyma.

(3) The frequency of coincident implantation metastases in the ureter or bladder or both.

The papillary epithelioma appears as a warty or villous proliferation of the pelvic mucosa. It invades the submucosa early and produces areas of ulceration.

Non-papillary carcinomas of renal pelvis are not as common as papillary tumors, but are met with often enough to be of clinical importance. The usual tumor in the small group is squamous cell carcinoma which is highly malignant with insidious onset and rapid and fatal course. This extremely malignant growth may spread by direct extension into the vena cava and to regional lymph nodes with metastases, also, to lungs, peritoneum and vertebrae. The prognosis is extremely grave. Kutzman has emphasized the importance

*Presented at American Urological Association, South Central Section, Denver.

of leukoplakia as a precursor of squamous cell carcinoma of the renal pelvis.

Etiology

Infection and stone may possibly have some part in the causation of the non-papillary neoplasms. Calculous disease is less commonly associated with the papillary growths.

Irritation, and infection producing leukoplakial and metaplasia changes in the mucosa are thought to play a definite part in the causation of the squamous-cell type of carcinoma. The importance of chronic irritation, inflammation and metaplasia in the pathogenesis of this disease is generally recognized. The rare mixed tumors are presumably embryonic in origin.

In the 19th century it was discovered that workers in the dye industries frequently developed bladder tumors, and since that time a great deal of study has been given to this subject. Tumors have been produced experimentally, in the pelvis, ureter and bladder of animals by using various carcinogenic agents.

It is presumed that the epithelium of the pelves, ureters and bladder has undergone premalignant changes from the effects of the carcinogens and that unrelated tumors have their origin simultaneously or successively in this altered epithelium. This presumption is adherred to by some investigators as well as the theory of implants of tumor particles transported by the urine.

Symptoms

Hematuria is the cardinal symptom of renal pelvic tumors because they are extremely vascular consisting principally of thin-walled blood vessels. The bleeding may be intermittent and painless at first, and very profuse later when the growths are more developed. Lumbar and abdominal pain may be mentioned by the patient, and also dysuria and frequency. Sometimes an abdominal mass will be noticed, usually from massive hydronephrosis which has been produced by the tumor obstructing the outlet of the pelvis.

Fever due to coexistent pyelitis and pyelonephritis may be a prominent feature. Occasionally there may be persistent digestive disturbances, presumably, from the close association of the sympathetic and parasympathetic nerve supply of the kidney and bowel.

Diagnosis

The diagnosis of renal pelvic tumors is made chiefly of urographic study. Intravenous urography may be of assistance but the information obtained is seldom as clear cut as by retrograde pyelography.

Bloody urine may be seen escaping from the ureteral orifice. Renal function may or may not be impaired.

Hydronephrosis may be present from the tumor growth obstructing the uretero-pelvic juncture and if the obstruction is complete, an enlarged kidney mass may be palpable.

In about 20 per cent of the cases cystoscopy will disclose an extension of the lesion by the presence of a papilloma of the bladder. This is usually located near the ureteral orifice of the involved kidney, but it may be located elsewhere in the bladder. Every case of bladder papilloma warrants a complete investigation of the upper urinary tract.

And it should be borne in mind that many instances of fatalities attributed to vesical malignancy would be shown to be directly due to the obstruction produced by papillary pelvic neoplasm, rather than to the influence of vesical tumor growths.

The pyelogram will show an irregular filling defect of the pelvis or calyx and often the pelvis or calyx will be dilated with a bizarre deformity. A complete outline of the ureter should be obtained, if possible. The pelvis may be filled with blood clots producing the characteristic pyelogram of this condition.

Cells from the urine should be stained by the Papanicolaou method, if they are present. Occasionally one may be able to obtain isolated tumor cells and possibly clumps of cells in ureteral catheter drainage when papillary tumors of the renal pelvis are present.

Prognosis

The prognosis of non-papillary tumors is extremely poor. Squamous-cell tumors have a very high mortality. Those who survive surgery usually are dead in less than a year. There are no five year cures on record. Unfortunately most of these patients are not seen by their physician until the growths have extended beyond the hope of cure.

In papillary tumors the prognosis is more favorable. They are less malignant, run a slower clinical course and if not too far advanced there

is better hope for a cure if the patient is seen soon enough and prompt surgery is performed.

Treatment

The best hope for cure lies in early diagnosis and prompt radical therapy (nephro-ureterectomy) whenever such is permitted by the condition of the patient.

Case One: M. M., male, age 88

This elderly man was admitted to the hospital suffering intense pain from an acute retention. The bladder was markedly distended.

One quart of bloody urine was withdrawn upon catheterization and later a considerable amount of blood clots were evacuated transurethrally. Active bleeding was seen to be issuing from the right ureteral orifice. The prostate was playing no part in the urinary difficulty as the vesical neck was not obstructed.

A retrograde pyelogram of the right kidney revealed a bizarre and mottled filling defect of pelvis and calices indicative of blood clots.

Figure 1

Pyelogram of right kidney showing mottled filling defect of pelvis and calices indicative of blood clots.

Due to the advanced age of this patient and his rather poor general condition, surgery was deferred. But despite repeated transfusions, the active bleeding from the right kidney persisted. Consequently, surgery was advised without further delay.

Operation—September 22, 1954: Under spinal anesthesia the kidney was exposed and the pelvis and upper ureter were observed to be markedly distended with blood clots. The kidney and ureter were removed.

Pathological Report

Macroscopic Examination:

This specimen is a complete right kidney with ureter attached. The kidney measures 13 cm. in length. The renal pelvis is markedly dilated. The capsule of the kidney is stripped. The surface is smooth, pale-grey and shows a number of tiny hemorrhages. Upon incision there is marked atrophy of the renal cortex. The renal pelvis and calyces are tremendously dilated and filled with hemorrhagic material. As this hemorrhagic material is removed, one sees that the surface of the renal pelvis and of the calyces is studded with innumerable extremely tiny pale-grey papillary structures which project into the lumen and measure up to 1 cm. in greatest diameter. Many of these papillary accumulations are friable and extremely hemorrhagic.

Microscopic examination:

Multiple sections are taken from various parts of the kidney and the ureter, as well as from the necrotic material contained in the renal pelvis. They show that the entire renal pelvis as well as the ureter is lined with innumerable papillary tumors of the transitional cell type. The individual stalks vary in size and depth of tissue. Sometimes a few thin layers are present. Sometimes the layers are 20 or 30 rows of cells deep. The individual tumor cells are fairly well differentiated. A certain irregularity of pattern is noticeable and a considerable number of mitotic figures are seen. In all the sections the tumor has remained superficial and does not invade the renal parenchyma.

Pathological Diagnosis:

Papillary transitional cell carcinoma of renal pelvis.

The post-operative convalescence was very satis-

factory and he left the hospital 12 days later. He was not seen after that time but his family reported a few months later that he was getting along satisfactorily.

It was learned later that he died, about one year after the operation, from "old age."

Case Two: M. F., male, age 58

This patient complained of pain in the left kidney area associated with hematuria. The symptoms had been present for eight days. Intravenous urograms by his family physician revealed no secretion of dye by the left kidney. The right kidney appeared normal in these films. The urine was bloody, resembling port wine.

Upon cystoscopy the drippings from the right kidney were clear whereas blood clots were observed to be exuding from the left orifice.

Retrograde pyelograms revealed the left pelvis to be filled with blood clots. The right kidney was normal in appearance. Operation was advised.

Figure 2

Pyelogram of left kidney showing pelvis filled with blood clots.

Operation—September 27, 1956: Under spinal anesthesia, the kidney was exposed and the pelvis was seen to contain a tumor-like mass with blood clots. The kidney and ureter were removed.

Pathological Report

Macroscopic Examination:

The specimen consists of a left kidney and ureter. The kidney measures 12 cm. from pole to pole and weighs 168.8 grams. The capsule of the kidney strips with ease. The renal surface is pale gray and dotted with numerous hemorrhagic spots. The cut surface of the kidney shows a usual renal pattern. Within the renal pelvis there is a coin-like plaque which measures 3 cm. in diameter. Numerous sections are taken through the plaque. On sectioning, this plaque is composed of firm grayish-white tissue with areas of necrosis in the center.

Microscopic Examination:

Multiple sections are taken from the plaque in the renal pelvis. The plaque shows large sheets of tumor cells which are epithelial in origin.

The individual tumor cells have elongated nuclei, which are hyperchromatic and show frequent pathologic mitotic figures. The tumor cells are arranged in a transitional cell pattern. They are forming solid sheets with areas of necrosis in the center. The tumor shows diffused and wide spread infiltrative invasion. The remainder of the kidney is not remarkable.

Pathological diagnosis:

Transitional cell carcinoma of renal pelvis (infiltrative).

The post-operative course for six months was very satisfactory, during which time he returned to work and had no complaint. The urine was clear. At this time, however, he began to have backache and general malaise. His condition became progressively worse and he died from generalized metastases nine months after the operation.

Case Three: W. H. S., male, age 74

In 1945, when he was 61 years of age, this patient first presented himself. He complained of dysuria, frequency and slight hematuria.

Cystoscopy revealed the presence of a small papilloma on the right trigone. Operation was refused and he was not seen again until 1948, three years later, when the hematuria had increased in severity and was giving him increased concern. By now the trigonal tumor had grown and was approximately the size of a pecan. This

tumor was resected and dessicated through a cystostomy incision, under spinal anesthesia.

In 1956, eight years later, hematuria returned and four small papillomas were seen on the bladder floor. These were resected and fulgerated, transurethrally.

In 1957 another episode of hematuria was experienced and upon cystoscopy recurrent trigonal papillary tumors were observed. One of them was located just within the right ureteral orifice. All of these small tumors were fulgerated, cystoscopically.

Six months later when bloody urine returned once more the bleeding was observed to be issuing from the right ureteral orifice. Retrograde pyelography now revealed blood clots in the pelvis of the right kidney. Intravenous urograms on several previous occasions had failed to give significant findings. Nephrectomy was advised.

Figure 3

Pyelo-ureterogram showing mottled appearance of pelvis and ureter from presence of tumor and blood clots.

Operation—June 12, 1957: Under spinal anesthesia, the right kidney was exposed and the pelvis and upper ureter were observed to be filled with clots. Complete nephro-ureterectomy was performed.

Pathological Report

Macroscopic Examination:

The specimen consists of the right kidney and the attached entire ureter, weighing together 107 grams. The kidney measures 10 x 6 x 3 cm. The ureter measures 29 cm. in length and ranges in diameter from 0.5 to 2 cm. The capsule of the kidney strips with marked resistance from a pinkish-gray surface showing large flattened scars.

The largest scar measures 5 cm. in diameter and is about 3 mm. deep. On cut surface the cortico-medullary markings are obscure. The cortex is thin measuring in places only 2 mm. in thickness. There is an increased amount of fat in the hilus of the kidney. The renal pelvis is dilated and bulges from the hilus.

When the ureter and renal pelvis are opened, a polypoid tumor mass is found in the pelvis. It measures 2.5 x 2 x 2 cm. Nearby are multiple smaller polypoid tumor masses ranging in size from 2 to 7 mm. in diameter. Similar tumor masses are present in the ureter all along its length. There is no invasion of the external surface of the ureter or renal pelvis. No tumor masses are found in the renal parenchyma, grossly. The renal vein and artery are not invaded by tumor tissue.

Microscopic Examination:

Sections of the tumor of the renal pelvis show thin papilliferous stalks covered by masses of atypical epithelial cells in several layers. These cells are small in size and resemble the transitional cell variety. They have a moderate amount of eosinophilic cytoplasm and relatively large hyperchromatic nuclei. Among the cells are some in mitosis. There is no invasion of the fibrous tissue of the renal pelvis and the tumor grows on the surface. Sections taken from the tumor nodules described grossly in the ureter show essentially similar tumor tissue to that described above. In places there is invasion of the submucosal connective tissue but no carcinoma cells are present in the muscularis. Sections of the kidney show severe chronic pyelonephritis but no invasion by tumor tissue.

Pathological diagnosis:

Papillary transitional cell carcinoma of renal pelvis with extension into the ureter and producing hydronephrosis.

The convalescence following this operation was very satisfactory and he remained well and returned to work for one year, when he experienced bloody urine once more. A small recurrent papilloma at the vesical neck was the source of the bleeding and this was thoroughly fulgerated, cystoscopically.

He has remained completely well since that time and the urine has remained clear. Two years have now elapsed since the nephro-ureterectomy.

Case Four: R. O., male, age 55

This patient complained of pain in the right loin associated with hematuria. The voided urine was very bloody. He was extremely tender over the right kidney.

Upon cystoscopy the urine from the right kidney was bloody. Bilateral pyelograms revealed a normal left kidney, whereas the right kidney was a huge hydronephrotic sac. 50 c.c. of opague media were employed in making the pyelogram. The left kidney had good function, but there was none in the right kidney. Operation was advised.

Figure 4

Pyelogram showing huge, hydronephrotic, functionless right kidney produced by complete obstruction by U.P.J. by renal pelvis tumor.

Operation—August 29, 1956: Under spinal anesthesia, the kidney was exposed and it was found to be a huge hydronephrotic mass. The pelvis appeared to be obstructed by a soft tumor mass at the uretero-pelvic juncture. The kidney and upper ureter were removed. It had been planned to free the lower ureter through a midline incision and perform a complete nephro-ureterectomy. However, due to the condition of the patient and advice of the anesthetist this latter procedure was postponed, and the nephrectomy wound was closed as quickly as possible. Later, he refused to have the lower ureter removed, and left the hospital.

Pathological Report

Macroscopic Examination:

The specimen consists of the right kidney and ureter. The kidney weighs 368 grams and measures 16 cm. in greatest diameter. The capsule of the kidney is dull and hyperemic. The kidney is of cystic consistency. On sectioning, the kidney is filled with purulent material. The renal pelvis is dilated and the renal cortex is nearly atrophic. Some of the calicies are covered with a rough partially necrotic membrane.

The renal pelvis is occluded by a large soft papillary tumor which is oval in shape and measures 4 cm. in greatest diameter. The tumor blocks the ureter completely. Sections are taken from the tip and from the base of the tumor. Several smaller tumors are present somewhat distal to the large tumor. In addition, the perirenal fat tissue is received. It is indurated and hyperemic.

Microscopic Examination:

The section from the renal cortex shows considerable atrophy and an acute inflammatory process. The interstitial tissue is heavily infiltrated with lymphocytes and plasma cells and in many areas, there is formation of young granulation tissue. The section from the renal pelvis shows a papillary tumor composed of sheets of moderately

anaplastic transitional cells. There is some invasion of the underlying stalk.

Pathological Diagnosis:

Papillary transitional cell carcinoma of renal pelvis with complete obstruction and massive hydronephrosis.

For one year he enjoyed good health and then after two episodes of hematuria from papillary tumor recurrence on the right trigonal area, he consented to the ureterectomy, which had been urgently advised.

Operation—January 17, 1959: Under spinal anesthesia the remaining portion of the right ureter was removed with a cuff of bladder. It was enlarged and distended with tumor growth.

Pathological Report

Macroscopic Examination:

The specimen is a long cylindric object 16 cm. from end to end, and 32 mm. in circumference in the opened condition. Externally this is grossly certainly compatible with being a greatly dilated ureter, having a membranous exterior to which some bits of fat are adherent, but the opened surface is completely filled from end to end, (with a tiny length of about 9 mm. of smooth tissue at one end, but no similar tumor free zone at the other) by a remarkable amount of papillary grey-pink tissue thrown up into numerous irregular little excrescencies. No zone of completely normal ureteral lining is visible. The tumor, even as opened and laid out, still attains a maximum thickness of 9 mm.

Microscopic Examination:

The tissue from the ureter shows an exophytic papillary transitional cell tumor, not noteworthily invading the substance of the ureteral wall itself anywhere. Although the cell sheets are becoming quite thick in places, and in these places sometimes an almost squamous form of the cell is seen, for the most part they retain a distinct long axis and reasonably good polarity, and form heavy caps on the extremely delicate stromal remifications.

The nuclei, for urinary tract epithelium, show moderate intra-series variation, and many of them are enlarged, more rounded than they should be, and show a visible but not prominent pink nucleolus. The mitotic rate is not high. The section cut from the one ureter shows superficial necrosis, with hemorrhage in the wall, but no tumor in the wall at either end (or for that matter in the middle) of the specimen. There is also a reactive sub-acute inflammation near the necrotic tissue.

Diagnosis:

Papillary transitional cell carcinoma, in right ureter.

A year has elapsed since the last operation and at the present time he is working regularly, with clear urine and no complaint.

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